SCHIZOPHRENIA
THE ADRENOCHROME HYPOTHESIS

The triggers that increase the negative impacts of genetic aberrations linked to schizophrenia have become increasingly more common in the industrialised world, yet most sufferers of this illness can be helped with an eight-step program.

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But strange that I was not told
That the brain can hold
In a tiny ivory cell
God's heaven and hell.

— Oscar Wilde (1854–1900)

THE WRONG ANSWER

Pope John Paul II wrote in a letter in 2002 that "It may be said that a society shows itself just to the extent that it meets the needs of all its members, and the quality of its civilisation is determined by the way in which it protects its weakest members".[1] If this is the case, then we should be judged harshly. In most "civilised" societies the mentally ill, mainly consisting of schizophrenics, are homeless, sleep in parks, under bridges or in dumpsters, beg muttering in the streets, or, because of their irrational crimes, fill our prisons.

Schizophrenia may be the cruellest disorder, afflicting "…young adults, often beginning insidiously and progressing until the ambitions, potentials, and hopes of early years are disregarded in disarray. In their place lie broken thoughts, inappropriate or stunted emotions, and internal voices or other misperceptions that can make existence a living hell."[2] Unfortunately, it is not rare.

Schizophrenia is the commonest serious mental illness of the developed world. In the USA, it accounts for some 24 per cent of all admissions to mental hospitals.[3] Initially, the disease is often episodic with acute phases interspaced with remissions, but it often becomes chronic.

In his extremely interesting book, *The Madness of Adam and Eve*, Horrobin[4] makes this point:

> *While in familial and personality terms the problem is devastating, in biochemical terms the problem cannot be very serious. After all, the young person functioned near normally for fifteen, twenty-five or thirty-five years before becoming ill. Moreover, all schizophrenic patients vary in the severity of their illness, often, as documented earlier, becoming near normal while the body temperature is elevated. The fundamental biochemical problem, therefore, cannot be too serious and must be reversible.*

This is an extremely intelligent and encouraging characterisation. It seems fair to ask, however, if the problem is so biochemically simple, why have countless thousands of doctors and scientists spent billions of dollars over more than 200 years in endless, unsuccessful attempts to discover the aetiology of schizophrenia?

The logical answer to this question must be that they are trying to hammer jigsaw puzzle pieces into spaces where they do not fit. Conventional drug treatment rests, to a large degree, on the "dopamine hypothesis"; that is, on the belief that excess dopamine accentuates and decreased dopamine reduces the positive or hot symptoms of schizophrenia.[5]

The evidence for high levels of dopamine in schizophrenia sufferers is poor,[6] and Parkinsonism (the mimicking of Parkinson's disease) has occurred frequently in patients treated for this hypothesised excess. Since Parkinson's disease is known to stem from a dopamine deficiency, it seems likely that drugs causing a similar illness in schizophrenics are creating a lack of this neurotransmitter rather than correcting an excess of it.

GENETIC PREDISPOSITION

If too much dopamine is not the root cause of schizophrenia, then what is? Certainly, genetics must play a significant role because 50 per cent of patients with this illness come from families with a history of the disorder.[7] This preponderance cannot be explained by abnormal child-rearing, since adoption has no impact on the risk of subsequently becoming schizophrenic.

There is beyond a doubt, therefore, a strong genetic component to schizophrenia, but it cannot be as straightforward as the inheritance of certain characteristics such as eye colour, since, as Myers[8] points out, "about half of the twins who share identical genes with a schizophrenic victim do *not* develop the disorder". One must agree with Nicol and Gottesman's[9] assessment that some individuals "have a genetic predisposition to the disorder but that this predisposition by itself is not sufficient for the development of schizophrenia." The schizophrenia genes, therefore, are not destiny but do enhance risk.

For many years, geneticists have been studying groups of schizophrenics or individual families in which the disorder is unusually common, in efforts to discover what, if any, genetic abnormalities they carry. To date, four genetic aberrations have been identified[10–13] that seem to occur more frequently than normal in subgroups of schizophrenics. These include the low enzyme activity variant of the catechol-O-methyltransferase gene, the GSTM1*O allele (required to produce a form of glutathione S-transferase) and possibly the C677TT variant of the gene for methylenetetrahydrofolate reductase. Beyond this, many schizophrenics appear to have inherited an unusual Nogo (reticulon 4, RTN4 or RTN-X) variant gene from both parents.

"We are now in the midst of an epidemic of insanity so insidious that most people are even unaware of its existence."

What these four genetic aberrations seem to have in common is that they all result in higher-than-normal exposure to adrenochrome, a metabolite of adrenaline, or in an abnormal susceptibility to its negative impacts. Since all of these variants are quite widely distributed in the human populace, it seems likely that abnormal levels of adrenochrome carry evolutionary advantages.

In several publications, including *What Really Causes Schizophrenia*,[14–16] I have argued that there appear to be not one but at least three and maybe four balanced genetic morphisms involved in this mental illness. The genetic aberrations increasing the risk of schizophrenia appear to promote religious sense, technical and artistic creativity, and leadership.[17] They also seem to provide a greater resistance to a wide range of cancers, especially that of the lung.[18] While they may be extremely destructive in individuals prone to develop schizophrenia, such genes are highly beneficial for humanity as a whole.

THE NATURE OF ADRENOCHROME

In the early 1950s, Osmond and Smythies[19] realised that pink (that is, deteriorated) adrenaline sprays were making some asthmatics psychotic, causing them to hallucinate.

Hoffer[20] knew that similar side effects accompanied the use of mescaline and made a list of all identified compounds that caused hallucinations in those who were awake. The list is short. It includes harmline, mescaline, ibogaine, d-lysergic acid diethylamide (d-LSD-25) and deteriorated adrenaline. Hoffer was delighted to realise that all were indoles (harmline, ibogaine, LSD) or could become indoles (mescaline). However, it was not known what pink adrenaline was until 1952, when Hutcheon[21] described how the oxidation of adrenaline created the indole adrenochrome. Hoffer then experimented by taking this substance himself, finding it made him paranoid.

In another trial, Osmond[22] was administered 300 mg of spray containing adrenochrome. Within 11 minutes, his ears felt plugged and his vision became abnormal. Rapidly swinging one arm backward and forward caused him to see this as a series of stationary arms. Within an hour, he decided to cycle home from the hospital and noticed that the roadside trees were expanding as if being pumped up with air. Clearly, he was hallucinating. Arriving home earlier than usual, he found his wife was out and he became very depressed, deciding that she must have left him and returned to her mother in a distant city. He counted all the suitcases. One was missing, increasing his certainty that she had gone. Finding a pile of clothes, he concluded that his wife had been packing to leave and had decided to go out to purchase her airline ticket. His depression increased. Remembering the experiment, he became very angry with the person who had forced him to take adrenochrome. (He had, in fact, been a very willing participant in the experiment.) Clearly, he was becoming paranoid.

This experimental evidence suggests that intelligent, highly educated individuals can be made to display many of the symptoms of acute schizophrenia very quickly, simply by exposing them to excess adrenochrome.

Put very simply, some people appear to become schizophrenic because their bodies manufacture an indole, adrenochrome, that has effects rather like the well-known psychedelic drug, LSD.

HISTORY OF SCHIZOPHRENIA

In a recently published book, *The Invisible Plague: The Rise of Mental Illness from 1750 to the Present*, Torrey and Miller[23] argue that, throughout human history, the baseline rate of insanity was approximately one case for each 2,000 members of society. Using a great diversity of records, ranging from mental health surveys to psychiatrists' diaries, they are able to prove beyond reasonable doubt that industrialisation has been accompanied by dramatic increases in mental illness. In England, Ireland, Canada and the USA, for example, "the prevalence of insanity, as a rate per population, increased at least sevenfold between the mid-18th and mid-20th centuries". In the USA and especially in Ireland, the increase was greater.

Torrey and Miller argue that "We are now in the midst of an epidemic of insanity so insidious that most people are even unaware of its existence". The invisible plague appears worst in Ireland, where the number of insane persons per 1,000 population has reached almost 8.0. This seems to be about 16 times the pre-industrial global baseline.

One does not have epidemics of genetic diseases, simply because the human genome does not alter rapidly enough to cause them. The current epidemic of insanity, associated with both schizophrenia and bipolar disorder, that has developed over the past 250 years is a very strong argument that the triggers that increase the negative impacts of the genetic aberrations linked to this mental illness have become more common. These appear to include anything that either stimulates the body's production of adrenaline or promotes its metabolism to adrenochrome and its derivatives.

PULLING THE TRIGGER

• Stress

Stress is the easiest way to promote the metabolism of adrenaline in the human body. Although medical interest in stress can be traced back to Hippocrates,[24] it was not until the 1920s that physiologist Walter Cannon[25] confirmed that response to stress is part of a unified mind-body system. Cannon was able to show that various stressors, including extreme cold, lack of oxygen and emotion-arousing incidents, trigger an outpouring of epinephrine (adrenaline) and norepinephrine (noradrenaline). These enter the bloodstream from sympathetic nerve endings in the inner adrenal glands.[26] In those stressed, the sympathetic nervous system increases respiration and heart rate, diverts blood to skeletal muscles and releases fat from storage. All these changes prepare the body for what Cannon called "fight or flight" and are obviously part of a response system that has evolved in an effort to deal with perceived threats.

Unfortunately, in situations of chronic stress, the "fight or flight" response becomes counterproductive, leading to a cumulative build-up of adrenaline, noradrenaline and cortisol. If these substances are not properly metabolised, long-term stress appears to promote disorders ranging from headaches and high blood pressure to rheumatoid arthritis and allergies.[27] What is significant here is that the "fight or flight" response to stress is associated with an elevation of adrenaline, oxidation of which can lead to an excess of adrenochrome. It is perhaps not surprising, then, that chronic stress is often linked to anxiety, poor concentration, depression, anger, frustration, fear and sadness.[28]

Of course, if the individual being stressed carries one of the genetic aberrations linked to schizophrenia, adrenochrome levels are likely to be higher than normal and may be linked to the paranoia and hallucinations that this indole causes when taken accidentally or experimentally.[29]

In summary, stress may be a trigger for schizophrenia because it increases the production of the precursors of adrenochrome.

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• Allergies

Physicians at the Moscow Psychiatric Institute used long fasts to treat schizophrenia, greatly improving the symptoms of 64 per cent of all chronic patients who completed their program.[30] This strongly suggests that there may be dietary triggers for the disorder. Further support for this possibility comes from the recognition that such fasting normalises catecholamine levels in the urine of schizophrenics.[31]

Countries where the national diet traditionally contains large quantities of cow's milk and wheat have poor recovery rates for schizophrenics.[32] This is to be expected, as some schizophrenics greatly improve on gluten-free diets,[33] perhaps because coeliac disease is common in their families.[34] Indeed, Pfeiffer claimed that 10 per cent of schizophrenics suffer from a gluten allergy.[35]

Hoffer also discovered that, in some fasting schizophrenics, the reintroduction of cow's milk caused hallucinations.[36] Indeed, 120 of Hoffer's "problem patients"—those who had not responded well to orthomolecular treatment—experienced significant permanent improvements in their mental health after identifying and eliminating from their diets specific foods to which they were allergic.[37]

These clues to the aetiology of schizophrenia suggest that diet often plays a key role in triggering the disorder. This may be one of the reasons why so many recovered schizophrenics believe they were formerly hypoglycaemic and that they had greatly improved only after a major dietary change. How can so many different foods trigger one disease? The best way to understand a disorder is often to examine extreme cases. A few people are exceedingly allergic to a particular food, such as peanuts or salmon, or to a product such as latex, and can die rapidly if exposed to even small quantities of it.[38–39] Allergic reactions can include skin rashes, itching, hives, burning eyes, swollen lips and tongue, difficulty breathing, wheezing, dizziness, abdominal pain, nausea and diarrhoea. In rarer cases, a strongly allergic individual suffers shock; blood pressure drops markedly, the throat swells and airways in the lungs constrict. Without immediate treatment with epinephrine, death from anaphylactic shock occurs.

Interestingly, the treatment of choice for anaphylaxis, whether caused by latex,[40] peanuts, or insect stings,[41] is always epinephrine, a dilute solution of adrenaline. This is because, during an allergic reaction, the chronic inflammatory response is usually characterised by numerous polymorphonuclear leukocytes,[42] the presence of which has been shown by Matthews and co-workers[43] to be linked to the oxidation of adrenaline to adrenochrome. In such an allergic reaction, oxidation of adrenaline to adrenochrome is detectable within five minutes and continues for at least four hours.

Of course, many people are allergic to substances that occur in water supplies or as air pollutants or as an integral part of products of one type or another.[44] This may be one of the reasons why schizophrenia's prevalence has markedly increased during the Industrial Revolution. Industrialisation has brought with it an enormous range of pollutants that have adversely affected air, water and soil quality. By 1977, the American Chemical Society had registered some four million chemical compounds, 32,000 of which were in commercial use.[45] It is unknown how many of these are potentially dangerous, although there are currently some 2,450 substances that are thought to cause cancer in the workplace. While attempts are generally made to establish the possible carcinogenicity of such industrial chemicals, their potential effects on mental health rarely appear to be considered.

• Hypoglycaemia

Hypoglycaemia was initially described by Dr Seale Harris[46] in 1924 when he discovered that sugar consumption stimulated the body to release insulin which, in turn, drove blood sugar levels down. Harris discovered that a high-protein, low-sugar diet—eaten at frequent, small meals—maintained a normal and stable blood sugar level, so controlling hypoglycaemia.

Since sugar consumption per capita in the United States has increased by roughly a factor of 20 since 1822,[47] hypoglycaemia has become rampant in its population.[48] As mentioned above, many recovered schizophrenics feel they had previously suffered from hypoglycaemia. Schauss[49] estimated that between 80 and 85 per cent of criminals in US prisons suffer from hypoglycaemia, often eating an excess of sugary foods and repeatedly drinking sugar-sweetened coffee and/or Kool-Aid.

It is well known that when blood sugar levels drop, adrenaline is released from the adrenal glands because it is involved in the metabolism of glucose.[50–51] It follows, therefore, that anyone suffering from the large blood-sugar swings characteristic of hypoglycaemia (associated with a diet that is too rich in sugar) is going to overproduce adrenaline. Hypoglycaemic individuals with one or more of the genetic aberrations seen in schizophrenia are therefore likely to suffer psychosis caused by adrenochrome created by the oxidation of this excess adrenaline.

BIOCHEMICAL AND CLINICAL IMPACTS OF ADRENOCHROME

Besides being an hallucinogen, adrenochrome is a highly reactive neurotoxin that, in schizophrenia, undermines at least three major biochemical systems.[52] It is an antagonist of the hormone triiodothyronine and can and often does seriously damage the thyroid. In chronic schizophrenics, this gland impairment appears to be permanent. Adrenochrome also has a Jekyll and Hyde relationship with serotonin and so impacts on tryptophan and its other chief metabolite, niacin. At low levels, serotonin appears to stimulate adrenochrome formation, while at higher levels it retards the process.

Adrenochrome also generates numerous free radicals, causing oxidative stress, eventually exhausting the schizophrenic antioxidant defence systems, creating deficiencies of glutathione peroxidase, superoxide dismutase and catalase. Complicating the impacts of high adrenochrome conversion from adrenaline are the numerous interactions that normally occur between triiodothyronine, serotonin and the three major components of the antioxidant defence system. In chronic schizophrenics who have suffered for years, these biochemical abnormalities result in brain atrophy, associated with large fluid-filled spaces known as ventricles, and serious damage to the thyroid gland.

Since many schizophrenics are overoxidising adrenaline due to allergic reactions, they need extra-special surroundings because of such sensitivities.

TREATMENT AND PREVENTION: EIGHT STEPS

If the adrenochrome hypothesis is correct, the "ideal" treatment for schizophrenia should involve eight steps, designed to reduce the production of adrenaline and slow down its metabolism to adrenochrome and other toxic indoles. Such a treatment should also attempt to reduce the further biochemical abnormalities that result from either an excess of adrenochrome and its metabolites or other impacts of the four genetic aberrations that appear associated with this mental illness.

Since many schizophrenics are overoxidising adrenaline due to allergic reactions, they need ***extra-special surroundings*** because of such sensitivities. Ideally, a treatment clinic would be like the Lange Meridian Centre, which was built using *Baubiologie* principles.[53] Step two involves ***genetic and biochemical screening*** to identify the most likely effective treatment protocol. ***Allergy testing*** is also essential, as is a ***low-sugar diet***.[54] The fifth step in the treatment of schizophrenia should involve ***medications that must quickly reduce the destructive impacts of excess adrenochrome and its derivatives***. They must also ***address the other biochemical anomalies directly related not to such indoles, but to the genetic aberration encouraging their overproduction***. In schizophrenics with the MTHFR C677TT variant, for example, the patient will also be suffering from depressed methionine and elevated homocysteine levels.

Endnotes

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There appear to be several avenues for lowering excess adrenochrome levels.[55] These include high doses of niacin or niacinamide and the use of other natural methyl acceptors such as thiamine (vitamin B1), riboflavin (vitamin B2) and ubiquinone (coenzyme Q10). Niacin is usually the treatment of choice.

Another adrenochrome antagonist, triiodothyronine, appears to be very effective in treating schizophrenia. As shown by Danziger,[56] every one of the 80 schizophrenics who had been ill for six months or less, who took between 120 and 1,200 milligrams of desiccated thyroid daily for at least 100 days, recovered, suffering relapses only if they later discontinued their medication. These doses may seem high, but it should be remembered that schizophrenics are known to be very resistant to thyroid medications. This is probably because all chronic schizophrenics appear to be suffering from badly damaged thyroid glands.

...all chronic schizophrenics appear to be suffering from badly damaged thyroid glands.

Treatment might also involve attempts to directly raise body levels of another adrenochrome antagonist, serotonin. If serotonin is not provided as a supplement, its metabolism could be encouraged by the consumption of foods that are high in tryptophan, such as beans, cod, pork, soybeans and cheese (provided that the patient is not allergic to them). In addition, every effort should be made to repair the antioxidant defence system, increasing glutathione peroxidase, catalase and superoxide dismutase activity.

Since there appear to be several genetic aberrations involved in schizophrenia, subgroups of patients also will suffer from *distinct biochemical imbalances that need correction*. The sixth step of the treatment protocol should address these. To illustrate, schizophrenics with the MTFR C677TT variant of the gene encoding for methylenetetrahydrofolate will suffer from an excess of homocysteine and a deficiency of methionine, even if treatment reduces adrenochrome levels. Beyond the provision of methionine, since the remethylation (or detoxification) of homocysteine requires folic acid, vitamin B12, zinc and trimethylglycine, it is likely that schizophrenics with this genetic aberration will require high doses of these nutrients.

Adrenochrome excess and the other biochemical abnormalities that occur in schizophrenia can eventually cause serious damage to the thyroid gland[57] as well as to the brain itself. Long-term chronic patients are therefore much more difficult to treat successfully. This task might not be impossible, but it will almost surely require higher doses of orthomolecular nutrients, taken for longer periods, before improvement is apparent.

As mentioned before, one of the major problems in chronic schizophrenia is the development of brain atrophy. Buckman and co-workers[58] provided evidence that blood levels of the selenoenzyme glutathione peroxidase have a strong negative correlation with computer tomography scan measures of such brain damage. Simply put, the less blood glutathione peroxidase, the greater the brain damage in chronic schizophrenics. Obviously, one treatment strategy worth trying is supplementation with the four nutrients—selenium, cysteine, glutamine and tryptophan—that the body requires to produce glutathione peroxidase.[59] Injected glutathione may be of value. There is also growing evidence that eicosapentaenoic acid can repair ventricle damage in chronic schizophrenics, leading to an improvement in their mental health.[60–62]

It is clear that damage is not restricted to the brain in chronic schizophrenics. All of these patients also appear to suffer from extensive thyroid abnormalities.[63] I do not know how to repair a damaged thyroid gland. If this is impossible, significant behavioural improvements can only be expected when using a protocol that includes continuous desiccated thyroid gland supplementation.

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The eighth and final step in the treatment of schizophrenia involves ***treatment for the soul***. Recovering schizophrenics are still one of the few groups society feels free to abuse, ostracise and discriminate against. While it is socially acceptable to admit to cancer, heart disease, multiple sclerosis or Parkinson's disease, admitting to schizophrenia invites fear and derision. To recover, schizophrenics need employment, respect and compassion. Too often, they receive rejection, abuse and insult. ∞

About the Author:

Harold D. Foster, PhD, was born and educated in England. He specialised in geology and geography, earning a BSc in 1964 from University College London and a PhD in 1968 from London University. He is a Canadian by choice, and has been a faculty member in the Department of Geography, University of Victoria, British Columbia, Canada, since 1967.

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He is also a member of the Science Advisory Panel for the Healthy Water Association.

Every day, Dr Foster makes a point of taking at least the recommended daily allowance of the known essential nutrients.

He is also currently pursuing offers for his suggested nutrient mixture to be produced for use in clinical trials with AIDS patients.

For a more detailed résumé, visit Dr Foster's website, http://www.hdfoster.com. Copies of his books, *The Ozymandias Principles*, *What Really Causes AIDS* and *What Really Causes Schizophrenia*, can be freely downloaded from his website.

To recover, schizophrenics need employment, respect and compassion. Too often, they receive rejection, abuse and insult.

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